L13 ANSWER 9 OF 1001 MEDLINE
AN 1998132976 MEDLINE
DN 98132976 PubMed ID: 9487008
TI **Neural stem** cells.
AU Murphy M; Reid K; Dutton R; Brooker G; Bartlett P F
CS Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital, Parkville, Victoria, Australia.
SO JOURNAL OF INVESTIGATIVE DERMATOLOGY. SYMPOSIUM PROCEEDINGS, **(1997 Aug)** 2 (1) 8-13. Ref: 58
Journal code: 9609059. ISSN: 1087-0024.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980319
Last Updated on STN: 19980319
Entered Medline: 19980310
AB This article is concerned with the idea that **neural precursor** cells in vertebrates can self-renew and give rise to all cell types within the nervous system. Supportive evidence for this notion of **neural stem** cells comes from clonal analyses undertaken both in vivo and in vitro. **Neural stem** cells also give rise to other cells in the body, including skin melanocytes and a range of mesenchymal cells in the head and neck. What determines the fate of these **stem** cells is their initial location within the developing **neural** tube and their final location post migration from the proliferative zone of the neural tube. A population of cells in the **adult** brain also have the characteristics of classical stem cells, a finding that opens the way for potential replacement therapy in nervous system-degenerative diseases. Much of the work in our laboratory has been concerned with the regulation of expansion and differentiation of these cells into their myriad progeny and the role of a series of various growth factors in this process. Different factors, such as members of the fibroblast growth factor family, act at different times to regulate stem cell proliferation and differentiation. Some factors, including members of the TGF beta superfamily, appear to be directly involved in the specification of cell fate. Finally, we are beginning to be able to determine the steps in the development of some lineages from multipotential stem cell to fully functional differentiated cell.

L17 ANSWER 34 OF 36 MEDLINE
AN 90278975 MEDLINE
DN 90278975 PubMed ID: 2112611
TI Fibroblast **growth factor** stimulates the proliferation and differentiation of **neural precursor** cells in vitro.
AU Murphy M; Drago J; Bartlett P F
CS Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital, Victoria, Australia.
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1990 Apr) 25 (4) 463-75.
Journal code: 7600111. ISSN: 0360-4012.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199007
ED Entered STN: 19900824
Last Updated on STN: 19900824
Entered Medline: 19900716
AB We have developed an in vitro culture system to study the regulation of proliferation and differentiation of neural precursor cells contained within the neuroepithelium of embryonic day 10 mice. A number of soluble **growth factors** have been tested for their ability to regulate these early events and, of these factors, we have found that the fibroblast **growth factors** [FGFs] can directly stimulate the proliferation and survival of the neuroepithelial cells. At least 50% of the neuroepithelial cells divide in the presence of FGF whereas in the absence of FGF all of the cells die within 6 days of culture. At higher concentrations of FGF, the cells change from being nonadherent round cells in tight clusters into a more flattened cell type which adheres to the substratum. This morphological change is accompanied by the expression of both neurofilament and GFAP, which are definitive markers of the two major cell types in the central nervous system: neurons and glia. In addition a neuroepithelial cell line, which does not rely on FGF for survival or proliferation, expresses both of these markers in response to FGF. These results indicate that FGF is stimulating the differentiation of the neuroepithelial cells into **mature neurons** and glia.

L19 ANSWER 30 OF 88 MEDLINE
AN 96158431 MEDLINE
DN 96158431 PubMed ID: 8594213
TI Neurotrophic factors in central nervous system trauma.
AU Mocchetti I; Wrathall J R
CS Department of Cell Biology, Georgetown University School of Medicine, Washington D.C. 20007, USA.
NC NS 01675 (NINDS)
NS28130 (NINDS)
NS32671 (NINDS)
SO JOURNAL OF NEUROTRAUMA, **(1995 Oct)** 12 (5) 853-70. Ref: 186
Journal code: 8811626. ISSN: 0897-7151.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199604
ED Entered STN: 19960422
Last Updated on STN: 19960422
Entered Medline: 19960409
AB Although regeneration of injured neurons does not occur after trauma in the central nervous system (CNS), there is often significant recovery of functional capacity with time. Little is currently known about the molecular basis for such recovery, but the increased trophic activity in injured CNS tissue and the known properties of neurotrophic factors in neuronal growth and maintenance suggest that these polypeptides are probably involved in recovery of function. Members of the neurotrophin family, including nerve growth factor (NGF), brain-derived neurotrophic factors (BDNF), and neurotrophin 3 (NT-3), are capable of supporting **survival** of injured CNS **neurons** both in vitro and in vivo. They also stimulate neurite outgrowth, needed for reorganization of the injured CNS, and the expression of key enzymes for neurotransmitter synthesis that may need to be upregulated to compensate for reduced innervation. The effects of the neurotrophins are mediated through specific high affinity trk receptors (trk A, B, C) as well as a common low affinity receptor designated p75NGFR. Another class of neurotrophic polypeptides also provides candidate recovery-promoting molecules, the heparin-binding growth factors' acidic and basic fibroblast growth factor (aFGF, bFGF). **FGFs** not only sustain **survival** of injured **neurons** but also stimulate revascularization and certain glial responses to injury. Both the neurotrophins and the **FGFs**, as well as their respective receptors, have been shown to be upregulated after experimental CNS injury. Further, administration of neurotrophins or **FGF** has been shown to reduce the effects of experimental injury induced by axotomy, excitotoxins, and certain other neurotoxins. The cellular basis for the potential therapeutic use of neurotrophic molecules is discussed as well as new strategies to increase neurotrophic activity after CNS trauma based on the recently obtained information on pharmacological and molecular control of the expression of these genes.

L17 ANSWER 33 OF 36 MEDLINE
AN 91043026 MEDLINE
DN 91043026 PubMed ID: 2172829
TI Proliferation and differentiation of **neuronal stem** cells regulated by nerve **growth factor**.
AU Cattaneo E; McKay R
CS Department of Brain and Cognitive, Massachusetts Institute of Technology, Cambridge 02139.
SO NATURE, (1990 Oct 25) 347 (6295) 762-5.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199012
ED Entered STN: 19910208
Last Updated on STN: 19910208
Entered Medline: 19901203
AB Nerve **growth factor** plays an important part in **neuron**-target interactions in the late embryonic and **adult** brain. We now report that this **growth factor** controls the proliferation of **neuronal precursors** in a defined culture system of cells derived from the early embryonic brain. Neuronal precursor cells were identified by expression of the intermediate filament protein nestin. These cells proliferate in response to nerve **growth factor** but only after they have been exposed to basic fibroblast **growth factor**. On withdrawal of nerve **growth factor**, the proliferative cells differentiate into neurons. Thus, in combination with other **growth factors**, nerve **growth factor** regulates the proliferation and terminal differentiation of **neuroepithelial stem** cells.

L9 ANSWER 3 OF 5 MEDLINE
AN 2002639722 MEDLINE
DN 22286075 PubMed ID: 12399108
TI **FGF-18** is a **neuron**-derived glial cell growth factor expressed in the rat brain during early postnatal development.
AU Hoshikawa Masamitsu; Yonamine Akiko; Konishi Morichika; Itoh Nobuyuki
CS Department of Genetic Biochemistry, Kyoto University Graduate School of Pharmaceutical Sciences, Yoshida-Shimoadachi, Sakyo, Kyoto 606-8501, Japan.
SO BRAIN RESEARCH. MOLECULAR BRAIN RESEARCH, (2002 Sep 30) 105 (1-2) 60-6. Journal code: 8908640. ISSN: 0169-328X.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200301
ED Entered STN: 20021026
Last Updated on STN: 20030123
Entered Medline: 20030122
AB We examined the expression of fibroblast growth factor-18 (**FGF-18**) in the rat brain during postnatal development by in situ hybridization. **FGF-18** was transiently expressed at the early postnatal stages in various regions of the rat brain including the cerebral cortex and hippocampus. **FGF-18** in the brain was preferentially expressed in **neurons** but not in glial cells. To elucidate the role of **FGF-18** in the brain, we examined the ligand-specificity of **FGF-18** by the BIAcore system. **FGF-18** was found to bind to FGF receptors (FGFRs)-3c and -2c but not to FGFR-1c, suggesting that **FGF-18** acts on glial cells but not on **neurons**. Therefore, we examined the mitogenic activity of **FGF-18** for cultured rat astrocytes and microglia. **FGF-18** was found to have mitogenic activity for both astrocytes and microglia. We also examined the **neurotrophic** activity of **FGF-18** for cultured rat cortical **neurons**. **FGF-18** was found to have no **neurotrophic** activity. The present findings indicated that **FGF-18** is a unique FGF that plays a role as a **neuron**-derived glial cell growth factor in early postnatal development when gliogenesis occurs.